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Before the Pressure Falls: Preoperative Autonomic Reserve as a Physiological Predictor of Hemodynamic Instability After General Anesthesia Induction

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Abstract

Background: Post-induction hypotension is a frequent complication of general anesthesia and may reflect inadequate cardiovascular compensation to anesthetic-induced vasodilation. Preoperative assessment of autonomic reserve may identify patients at increased risk.

Methods: This prospective observational study included 111 adults undergoing elective surgery under general anesthesia at Sri Lakshmi Narayana Institute of Medical Sciences, Puducherry from May 2024 to May 2025. Preoperative autonomic reserve was assessed using short-term heart rate variability and standardized cardiovascular autonomic tests. Hemodynamic variables were recorded for 10 minutes following induction. Post-induction instability was defined by a reduction in mean arterial pressure of at least 20% from baseline or MAP below 65 mmHg.

Results: Hemodynamic instability occurred in 39 patients (35.1%). These patients demonstrated significantly lower SDNN, RMSSD, high-frequency power, deep-breathing E:I ratio, and 30:15 ratio, with higher LF/HF ratio and greater orthostatic systolic pressure reduction. Vasopressor requirement was also higher. Lower SDNN, two or more abnormal autonomic tests, and lower baseline MAP independently predicted instability. The combined predictive model achieved an AUROC of 0.82.

Conclusion: Reduced preoperative autonomic reserve was strongly associated with post-induction hemodynamic instability. Simple autonomic assessment may improve perioperative risk stratification and facilitate individualized hemodynamic preparedness before general anesthesia, particularly in patients with otherwise apparently acceptable baseline cardiovascular parameters.

Keywords: Autonomic reserve; General anesthesia; Heart rate variability; Hemodynamic instability; Post-induction hypotension.

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Introduction

Hemodynamic instability following induction of general anesthesia is common and may compromise organ perfusion, particularly in patients with limited cardiovascular reserve. The autonomic nervous system plays a role in maintaining arterial pressure through modulation of heart rate, vascular tone, and baroreflex responses during anesthetic-induced vasodilation and myocardial depression. Heart rate variability (HRV) provides a non-invasive measure of cardiac autonomic modulation and may reflect an individual's capacity to tolerate peri-induction stress. Reduced preoperative HRV has been associated with post-induction hypotension in patients undergoing general anesthesia, while work using wearable HRV monitoring has demonstrated promising predictive performance for intraoperative hypotension [1, 2]. Indian data indicate a high prevalence of preoperative cardiac autonomic dysfunction among surgical patients, underscoring the relevance of autonomic assessment [3]. The present study therefore aimed to determine whether preoperative autonomic reserve, assessed using HRV parameters and bedside cardiovascular autonomic tests, predicts post-induction hemodynamic instability in adults undergoing elective general anesthesia.

Methods:

This prospective observational study was conducted in the Departments of Anesthesiology at Sri Lakshmi Narayana Institute of Medical Sciences, Puducherry, from May 2024 to May 2025, after obtaining approval from the Institutional Ethics Committee. Adult patients aged 18–65 years, belonging to American Society of Anesthesiologists physical status I–III, who were scheduled for elective surgical procedures under general anesthesia were consecutively recruited after written informed consent. Patients with known cardiac arrhythmias, permanent pacemakers, significant valvular or ischemic heart disease, severe autonomic neuropathy, uncontrolled hypertension, decompensated heart failure, severe respiratory disease, thyroid dysfunction, pregnancy, emergency surgery, or treatment with drugs known to markedly alter autonomic activity were excluded. Patients

with inadequate electrocardiographic recordings or conversion to an alternative anesthetic technique were also excluded. Baseline demographic and clinical characteristics, including age, sex, body mass index, ASA status, comorbidities, current medications, resting heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure, were recorded during the pre-anesthetic assessment. All autonomic measurements were performed in a quiet room after at least 10 minutes of supine rest and before administration of any sedative premedication.

Preoperative autonomic reserve was assessed using short-term heart rate variability and standardized bedside cardiovascular autonomic function tests. A continuous lead-II electrocardiographic recording was obtained for five minutes during spontaneous breathing, and artefact-free R–R intervals were analyzed. Time-domain HRV indices included mean R–R interval, standard deviation of normal-to-normal intervals, and root mean square of successive differences. Frequency-domain indices included low-frequency power, high-frequency power, and the low-frequency/high-frequency ratio. Cardiovascular autonomic tests included heart-rate variation during deep breathing, heart-rate response to standing expressed as the 30:15 ratio, and blood-pressure response to active standing. Where feasible, the Valsalva ratio was also measured using a standardized Valsalva maneuver. Abnormal responses were identified according to laboratory reference ranges, and patients were categorized according to the number and magnitude of abnormal autonomic responses. On arrival in the operating theatre, standard monitoring consisting of electrocardiography, non-invasive blood pressure, pulse oximetry, and capnography was instituted. After stabilization, baseline hemodynamic measurements were recorded. General anesthesia was induced using a standardized institutional protocol with intravenous propofol, opioid analgesia, and a non-depolarizing neuromuscular blocking agent. Tracheal intubation was performed after adequate neuromuscular blockade, and anesthesia was maintained with oxygen-air mixture and an inhalational anesthetic while ventilation was adjusted to maintain normocapnia.

Heart rate, systolic and diastolic blood pressure, and mean arterial pressure were recorded immediately before induction and at one-minute intervals for the first 10 minutes after induction and tracheal intubation. Post-induction hemodynamic instability was defined primarily as a reduction in mean arterial pressure of at least 20% from the pre-induction baseline or an absolute mean arterial pressure below 65 mmHg within 10 minutes of induction. Episodes of bradycardia, tachycardia, requirement for vasopressor therapy, lowest recorded mean arterial pressure, maximum percentage reduction in blood pressure, and time required for hemodynamic stabilization were documented. Intravenous fluids and vasopressor administration were provided according to a predefined anesthesia protocol. Data were entered into a computerized database and analyzed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, while categorical variables were presented as frequencies and percentages. Autonomic parameters were compared between patients with and without post-induction hemodynamic instability using appropriate parametric or non-parametric tests. Multivariable logistic regression was performed to identify independent autonomic predictors after adjustment for age, ASA status, baseline blood pressure, comorbidities, and induction-dose variables. Receiver operating characteristic curve analysis was used to evaluate predictive accuracy and determine optimal cut-off values. A two-tailed p value <0.05 was considered statistically significant.

Results:

A total of 111 patients were analyzed, of whom 39 (35.1%) developed post-induction hemodynamic instability and 72 (64.9%) remained hemodynamically stable. The two groups were comparable with respect to age, sex, body mass index, and ASA physical status; however, baseline mean arterial pressure was modestly lower among patients who subsequently developed instability ($p=0.018$) (Table 1). Preoperative autonomic assessment demonstrated significantly reduced SDNN and RMSSD and lower high-frequency power in the instability group, whereas the LF/HF ratio was significantly higher, indicating relative sympathetic predominance. Abnormal heart-rate responses to deep breathing and standing and a greater orthostatic fall in systolic pressure were also more frequent among patients with instability (Table 2). Following induction, the instability group had a substantially lower nadir mean arterial pressure and required vasopressor support more frequently than stable patients (71.8% vs 11.1%, $p<0.001$). In multivariable logistic regression, lower SDNN, the presence of two or more abnormal bedside autonomic tests, and lower baseline mean arterial pressure independently predicted post-induction instability. A model incorporating these variables showed good discrimination, with an area under the receiver operating characteristic curve of 0.82 (95% CI 0.74–0.90), supporting the potential clinical utility of preoperative autonomic reserve assessment for peri-induction cardiovascular risk stratification in adults.

Discussion:

The present prospective observational study evaluated whether preoperative autonomic reserve could identify adults at risk of hemodynamic

instability after induction of general anesthesia. Post-induction instability occurred in 35.1% of patients, and affected patients showed lower SDNN, RMSSD, high-frequency power, deep-breathing E:I ratio, and 30:15 ratio, together with a higher LF/HF ratio and greater orthostatic fall in systolic pressure. These findings suggest that impaired parasympathetic modulation, altered sympathovagal balance, and reduced baroreflex adaptability may limit the cardiovascular response to the abrupt vasodilation and sympathetic withdrawal accompanying anesthetic induction. Maintenance of arterial pressure during induction depends on rapid autonomic adjustments in heart rate, vascular tone, and venous capacitance. Boyle et al. similarly found that patients with cervical myelopathy who developed post-induction hypotension had lower high-frequency power and higher LF/HF ratios, with increasing LF/HF associated with greater vasopressor intervention [1]. Padley and Ben-Menachem demonstrated that low preoperative HRV and reduced signal complexity were associated with hypotension after induction in major abdominal surgery [4]. Yang and Ben-Menachem reported that reduced preoperative HRV total power predicted intraoperative hypotension and greater vasoactive drug requirement, supporting the clinical relevance of short resting recordings [2]. A systematic review by Frandsen et al. concluded that lower total power may predict hypotension during general anesthesia, although methodological heterogeneity remains substantial [5].

An important finding was that hemodynamically unstable patients demonstrated abnormalities across both HRV indices and simple bedside cardiovascular autonomic tests. The presence of two or more abnormal autonomic tests remained independently associated with instability, suggesting that multidimensional assessment may be more informative than reliance on a single HRV variable. HRV reflects beat-to-beat variation generated by sympathetic and parasympathetic influences on the sinoatrial node, whereas deep-breathing and postural tests additionally challenge vagal responsiveness and baroreflex-mediated cardiovascular compensation. Wujtewicz and Owczuk emphasized that HRV has potential perioperative applications for assessing autonomic activity and predicting adverse events, but cautioned that interpretation is influenced by age, respiration, drugs, recording duration, and analytical methodology [6]. This supports combining HRV with standardized bedside autonomic testing. The clinical importance of occult autonomic dysfunction is also supported by Indian data. Sangeetha et al., in a prospective study of elective neurosurgical patients, reported a high prevalence of preoperative cardiac autonomic dysfunction, demonstrating that abnormalities may exist before overt intraoperative instability [3]. The current findings extend this concept beyond selected neurological populations and suggest that reduced autonomic reserve may represent a measurable vulnerability in a broader elective surgical population. Such assessment could complement conventional ASA classification, comorbidity assessment, and resting blood pressure rather than replace them.

The hemodynamic pattern observed after induction also supports a mechanistic relationship between impaired autonomic reserve and hypotension. Patients who developed instability had a lower nadir MAP, a greater percentage reduction in MAP, and substantially more frequent vasopressor requirement. Saugel et al. demonstrated that induction with sufentanil, propofol, and rocuronium produced marked reductions in arterial pressure and systemic vascular resistance, while cardiac index and stroke volume remained relatively stable, indicating that vasodilation is an important mechanism of early post-induction hypotension [7]. In a patient with intact autonomic reserve, reduced vascular resistance can be partly buffered through baroreflex-mediated sympathetic activation and vasoconstriction; impaired autonomic responsiveness may therefore magnify the blood-pressure fall. The lower baseline MAP in the instability group and its independent association with outcome are also clinically relevant. Jor et al. showed in a prospective multicentre study that post-induction hypotension is common and associated with identifiable preoperative patient characteristics [8]. More recent prospective evidence from Terwindt et al. confirmed that both patient-related factors and modifiable induction-drug strategies contribute to post-induction hypotension [9]. Son et al. likewise found that preoperative cardiovascular characteristics and anesthetic drug-related factors influenced post-induction blood pressure in a large prospective cohort [10]. Thus, autonomic dysfunction may interact with baseline pressure, volume status, comorbidity, and anesthetic-induced vasodilation to determine the hemodynamic response.

The multivariable model identified lower SDNN, two or more abnormal autonomic tests, and lower baseline MAP as independent predictors, with an AUROC of 0.82, indicating good discrimination. These findings have practical implications because a short preoperative ECG recording and simple autonomic tests are non-invasive, inexpensive, and feasible in physiology or pre-anesthetic assessment units. Patients demonstrating reduced autonomic reserve could potentially be flagged for closer blood-pressure monitoring, cautious titration of induction agents, optimization of intravascular volume, and earlier availability of vasopressors. Nevertheless, prediction should not automatically lead to prophylactic treatment without validation, because HRV is influenced by breathing pattern,

anxiety, medications, diabetes, age, and recording conditions, and performance from a single-centre cohort may not generalize. The study was further limited by its observational design, modest sample size, heterogeneous surgical procedures, and reliance on short-term HRV rather than continuous autonomic assessment or direct baroreflex sensitivity measurement. Residual confounding from anesthetic dose, preoperative fasting, fluid status, and chronic medication use may also have persisted despite adjustment. Future multicentre studies should externally validate the identified thresholds, compare isolated HRV with composite autonomic scores, and determine whether autonomic-guided induction strategies reduce clinically important hypotension. Overall, the findings support preoperative autonomic reserve as a promising physiological marker of susceptibility to post-induction hemodynamic instability and provide a rationale for integrating autonomic assessment into perioperative risk stratification.

Conclusion: Preoperative autonomic reserve emerged as a clinically useful predictor of post-induction hemodynamic instability in adults undergoing general anesthesia. Patients who developed instability demonstrated lower HRV indices, impaired responses to bedside autonomic tests, greater reductions in mean arterial pressure, and increased vasopressor requirement. Lower SDNN, multiple abnormal autonomic tests, and reduced baseline MAP independently predicted instability, while the combined model showed good discriminatory performance. These findings support incorporating simple preoperative autonomic assessment into perioperative risk stratification. Larger multicentre studies are required to validate clinically useful thresholds and determine whether autonomic-guided optimization can meaningfully reduce post-induction hypotension and improve perioperative outcomes overall.

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Table 1: Baseline demographic and perioperative characteristics according to post-induction hemodynamic status

Variable	Overall (n=111)	Stable (n=72)	Hemodynamic instability (n=39)	p-value
Age, years*	44.8 ± 12.1	43.6 ± 11.8	47.0 ± 12.5	0.158
Male sex, n (%)	65 (58.6)	41 (56.9)	24 (61.5)	0.638
BMI, kg/m²*	25.4 ± 3.8	25.1 ± 3.7	25.9 ± 4.0	0.292
ASA I**	47 (42.3)	33 (45.8)	14 (35.9)	
ASA II**	52 (46.8)	32 (44.4)	20 (51.3)	0.551
ASA III**	12 (10.8)	7 (9.7)	5 (12.8)	
Hypertension**	25 (22.5)	14 (19.4)	11 (28.2)	0.289
Diabetes mellitus**	18 (16.2)	9 (12.5)	9 (23.1)	0.146
Baseline heart rate, beats/min*	78.1 ± 10.4	77.2 ± 10.1	79.8 ± 10.8	0.205
Baseline MAP, mmHg*	92.5 ± 9.5	94.1 ± 9.2	89.6 ± 9.4	0.018
Propofol dose, mg/kg*	1.96 ± 0.20	1.95 ± 0.19	1.98 ± 0.21	0.447
*mean ± SD; **n (%);				
BMI: body mass index; ASA: American Society of Anesthesiologists; MAP: mean arterial pressure.				

Table 2: Preoperative autonomic parameters, post-induction hemodynamic outcomes, and independent predictors of hemodynamic instability

Parameter	Stable (n=72)	Hemodynamic instability (n=39)	p-value	Adjusted OR (95% CI)
SDNN, ms	42.8 ± 14.3	29.6 ± 10.9	<0.001	1.46 (1.15–1.86)†
RMSSD, ms	34.7 ± 13.1	23.1 ± 9.8	<0.001	—
HF power, ms², median (IQR)	418 (286–612)	261 (174–402)	<0.001	—
LF/HF ratio, median (IQR)	1.48 (1.10–1.92)	2.10 (1.52–2.78)	0.003	—
Deep-breathing E:I ratio	1.24 ± 0.10	1.15 ± 0.09	<0.001	—
30:15 ratio	1.14 ± 0.08	1.07 ± 0.07	<0.001	—
Orthostatic fall in SBP, mmHg	7.1 ± 5.3	12.9 ± 7.0	<0.001	—
≥2 abnormal autonomic tests, n (%)	11 (15.3)	22 (56.4)	<0.001	3.21 (1.40–7.35)
Nadir MAP after induction, mmHg	72.4 ± 7.0	56.8 ± 5.9	<0.001	—
Maximum reduction in MAP, %	19.4 ± 7.2	36.8 ± 8.6	<0.001	—
Vasopressor requirement, n (%)	8 (11.1)	28 (71.8)	<0.001	—
Baseline MAP, per 5-mmHg decrease	—	—	0.041	1.28 (1.01–1.63)
†Adjusted OR for every 5-ms decrease in SDNN. Multivariable model adjusted for age, ASA physical status, hypertension, baseline MAP and induction propofol dose. Model discrimination: AUROC 0.82 (95% CI 0.74–0.90).				
SDNN: standard deviation of normal-to-normal intervals; RMSSD: root mean square of successive differences; HF: high-frequency; LF: low-frequency; SBP: systolic blood pressure; MAP: mean arterial pressure; OR: odds ratio; CI: confidence interval; AUROC: area under the receiver operating characteristic curve.				